**Entamoba histolytica (amebic dysentery)**

A 36-year old man presented to the ER with a 10-day history of intermittent **diarrhea and tenesmus (feeling of constantly needing to pass stool),** with **blood and mucus visible in the stool.** He had just returned from a working **trip to Indi**a, where he had **visited a rural town** in the last week of his trip.

* IMAGING
  + Sigmoidoscopic examination revealed multiple **small hemorrhagic areas with ulcers**
* DIAGNOSTIC WORK UP
  + Enteric (bacterial) cultures
  + Stool Ag test for amebic agent
  + Microscopic (ova and parasite) examination\*\* (recommended for parasite)
* DIFFERENTIAL
  + Dysentery syndrome due to Entamoeba histolytica, Enteroinvasive E. coli, Salmonella, Shigella, Yersinia enterocolitica
  + IBD
  + Rationale: Stool studies required for definitive diagnosis though history of exposure is helpful. E. histolytica and S. dysenteriae (bacillary dysentery) are two of the most common colonic ulcerative diseases but are much more common in developing countries (history therefore important). IBD considered after infectious etiologies are ruled out.
* MICROBIOLOGICAL PROPERTIES
  + Exist as trophozoite or a cyst
    - **Trophozoite (15-20 um) has a single nucleus** with a central karyosome and uniformly distributed peripheral chromatin. It cannot live outside host
    - **Cyst (12-15 um) is spherical with four nuclei** with central karyosomes and fine, uniformly distributed peripheral chromatin. They are hardy (resist environment and stomach pH), **Cysts = Infectious form.**
  + Ova and Parasite exam recommended for **light microscopy does NOT allow distinction between the invasive E. histolytica and lumen-dwelling E. dispar unless erythrophagocytosis** (the presence of ingested RBCs in trophozoites) can be demonstrated.
  + Must be distinguished from non-pathogenic amebae. Presence of non-pathogenic ameba in stool is strongly indicative of poor sanitation and is also a warning sign of possible pathogenic E. histolytica
* TRANSMISSION
  + Humans are natural reservoir. Cysts passed in human feces. Cysts are hardy and survive in environments. Infection occurs due to **ingestion of MATURE cysts in fecally contaminated food, water, or hands.**
* PATHOGENESIS
  + **Ingests infective** **cysts. Excystation** occurs in small intestine and **trophozoites released**. Trophozoites migrate to large intestine and adhere to intestinal mucosal cells via lectin-binding receptors. Trophozoites may remain as commensals in which trophozoites multiply by binary fission and produce cysts that are passed in feces.
  + In some, trophozoites invade the intestinal mucosa with virulence factors: cytotoxins and cysteine proteases.
    - **Cytotoxins** enable trophozoites to **invade the colon**, with **lysis of epithelial cells**. They can also **lyse PMNs,** releasing hydrolytic enzymes that contribute to damage.
    - **Cysteine proteinases degrade collagen and elastin**.
    - **Trophozoites feed on neutrophils, monocytes, and lymphocytes, RBS, and other host materials, causing amebic colitis.**
  + Lesions in the colon range from non-specific colitis with inflammatory cells and E. histolytica to **flask shaped-ulcers** and may extend thro tissue planes
  + In some, trophozoites may **invade the bloodstream** and reach extraintestinal sites such as the liver (where it can irreversibly destroy hepatocytes and form **amebic liver abscess**), brain, and lungs. Blood borne trophozoites are resistant to complement-mediated lysis.
* TREATMENT
  + **Metronidazole** for systemic therapy. For symptomatic patients, iodoquinol, paraomomycin or diloxanide is used in conjunction with metronidazole to treat intraluminal infection. This combo achieves COMPLETE elimination and cure.